

## Section of Pædiatrics

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### DISCUSSION ON TUBERCULOUS CERVICAL ADENITIS

Dr. F. J. W. Miller (Royal Victoria Infirmary, Newcastle upon Tyne):

#### *The Problems of Peripheral Tuberculous Lymphadenitis in Childhood*

I shall discuss only our own experience of this form of tuberculosis.

I shall consider not only cervical adenitis but also superficial or peripheral tuberculous adenitis in any part of the body, for it is all part of the same problem. My own interest arose as part of a wider concern with childhood tuberculosis and, in particular, from a study of the behaviour of glands in a group of children with visible primary infection on skin or mucosa (Miller and Cashman, 1955). This, in turn, broadened into a wider study of all the cases seen in our department in Newcastle over a period of seven years, made in an attempt to answer four major questions.

- (1) What is the origin of peripheral tuberculous lymphadenitis?
- (2) What is the natural history if untreated?
- (3) What is the variation of the clinical picture?
- (4) What is the most effective method of treatment?

Three basic conditions must be fulfilled before the natural history of tuberculosis can be studied.

- (i) The time, within defined limits, and site of primary infection must be known.
- (ii) A large group of children of all ages must be studied (ideally all the children in a population).
- (iii) The study must continue long enough to see the ultimate manifestations of clinical disease.

Very few studies of tuberculous lymphadenitis satisfy these conditions, and this is perhaps the major reason why our knowledge is so uncertain.

*How do peripheral tuberculous glands arise.*—We think that wherever this occurs in childhood, it is almost always the glandular component of a primary complex, either the primary glands themselves (the first affected glands) or glands farther down the same lymphatic chain. There are exceptions to this, but they are uncommon. Some of our evidence is given in Fig. 1, showing the distribution of adenitis in 168 children with peripheral lymphadenitis.

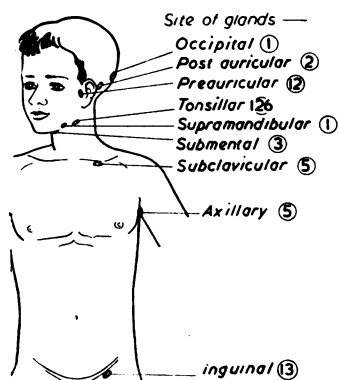


FIG. 1.—Distribution of peripheral tuberculous lymphadenitis in 168 children (Boyes *et al.*, 1956).

In 125 it was the *only* evidence of the primary infection and in another 33 there was, in addition, other evidence of primary calcification indicated by calcification of the root of the lung (17) or in the abdomen (16), including 2 with calcification in both chest and abdomen. In all cases other than those in the tonsillar or submandibular glands, we knew the precise site of the primary focus. But one of the real difficulties in detecting the primary focus in the oro-pharynx is that it is visible for only a limited time and may disappear without leaving

a scar. This phenomenon is clearly seen in Figs. 2 and 3, showing a permanent tooth erupting at the site of a primary focus in the crater of the preceding deciduous tooth. Thus, on clinical grounds, we feel that peripheral lymphadenitis is an expression of primary infection and therefore suggest that primary infection, outside the chest and abdomen, is much more common than we have formerly believed.



FIG. 2.—Circular ulcer at site of tooth extraction.

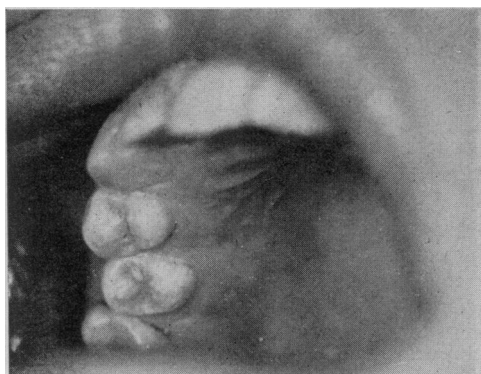


FIG. 3.—Same case as Fig. 2. Eighteen months after infection the site of primary infection has been obliterated by the permanent tooth (Boyce *et al.*, 1956).

Figs. 2 and 3 are from *Arch. Dis. Childh.* (1956) by kind permission.

*What is the natural history of peripheral lymphadenitis?*—We have found that the great majority of untreated peripheral tuberculous glands sooner or later soften and form abscesses which rupture spontaneously. Indeed, only 15 from 230 cases followed over a minimal period of two years did not give rise to abscess formation; sometimes softening did not occur for four to five years after infection. This tendency to soften should be the central fact in determining treatment; once softening and spontaneous rupture have occurred the period of illness is likely to be long.

If spontaneous softening has once occurred in a gland we found that in almost half the cases there was a further gland abscess either at the site of the primary gland or further down the same lymphatic chain.

Glands tend to soften after intercurrent infection, especially upper respiratory infections. This is not due to direct invasion of the gland by other organisms, for the pus obtained from the abscess is either sterile or contains only tubercle bacilli, but is due to a temporary diminution of the patient's resistance, possibly together with increased gland activity. For example, a child presented with simultaneous softening of the pre-auricular, tonsillar and post-sternomastoid glands three years after primary infection of skin of the forehead and immediately after measles.

The incidence of hæmatogenous complications in children with peripheral lymphadenitis is of the same order as in primary infection of chest or abdomen, but children with primary infection in the lungs or abdomen only very rarely get peripheral lymphadenitis as a result of hæmatogenous spread.

*Clinical picture of peripheral lymphadenitis.*—Two major groups of cases are seen:

- (i) Children with lymphadenitis after recent infection.
- (ii) Children with softening in glands infected more than one to two years previously.

Children with glands appearing at the time of primary infection present the same spectrum of reaction to primary infection as children with infection in the lungs or abdomen. There are only two differences; when the infection is in the lungs the glands are not visible, but on the other hand, the child with peripheral lymphadenitis escapes the danger of bronchial involvement. In the majority of children there is some diminution of zest and vigour with a slow progressive enlargement of the regional gland, which is clinically the largest gland, and its satellites, to form a group of smaller glands about the regional; sometimes these glands proceed slowly but inevitably to softening, in others they subside only to enlarge again some time thereafter.

In about one-tenth of the cases the reaction is much more acute; the child is sharply febrile with a temperature ranging from 101° to 104° F., he is highly sensitive to tuberculo-protein and may develop erythema nodosum. The glands enlarge much more rapidly and

become one mass: they may appear hot and rather tender but are never so tender as in acute pyococcal adenitis and do not restrict function. These glands soften very rapidly and even with chemotherapy abscess formation occurs within a month or so.

*Softening in old glands.*—If softening occurs in glands infected a year or more earlier, calcification is often visible on radiological examination. The child is not so well as usual, but acute symptoms or more severe disturbances, and the acute reaction described above, do not occur. The softening is usually rapid and the skin may be involved in two to three weeks once the abscess has become apparent.

*What is the most effective treatment of tuberculous lymphadenitis?*—The objects of treatment can be described as follows:

- (a) To avoid abscess formation and skin involvement.
- (b) To avoid a long period of ill-health or hospital treatment.
- (c) To obtain a good cosmetic result.

If the glands are seen shortly after infection and before they soften, it is usually possible to attempt all three objectives, but often softening is present before the child is seen and then only the second and third of these criteria can be pursued.

I have no experience with calciferol or with X-ray therapy and shall not consider these methods here.

*Effect of chemotherapy on glands.*—Can glands which are firm when first seen be prevented from softening by chemotherapy alone? We found that with an initial course of streptomycin and PAS for three to four weeks the glands first become smaller and more discrete, but once the chemotherapy has been stopped the glands sooner or later have softened. Since the introduction of isoniazid and the use of longer courses of therapy, we still do not know if isoniazid and PAS, given over long periods, will prevent softening until firm calcification has occurred. I have a small group of children on such treatment, but it will be at least two to three years before I get the answer.

With our present knowledge of the natural history of tuberculous lymphadenitis and of the action of antibiotics, we must rely upon a combination of surgery and antibiotics.

The best results are achieved when the glands are firm and recent when first seen and the child has not had a marked febrile reaction. If streptomycin and isoniazid are given there is both a general and local effect. The general condition improves rapidly and at the same time the gland mass shrinks and sometimes resolves into separate discrete glands. This reduction in size will continue for about three weeks, but the glands do not return to normal and usually one remains larger than the others. I believe, subject only to the possible effect of long-term isoniazid and PAS, that the correct advice at this stage is to remove by *local* dissection 2 or 3 of the largest glands, but not with a block dissection. A planned incision gives a good cosmetic result and none of the 20 cases we have done at this stage have had subsequent abscesses. The antibiotics should be continued over the period of operation and then isoniazid and PAS given for three months afterwards.

*Recent infection with a marked febrile reaction.*—This reaction is uncommon but dramatic. The effect of streptomycin and isoniazid is also striking, the temperature falls and the gland then gets smaller rapidly (Fig. 4). But softening also occurs rapidly in two to three weeks,

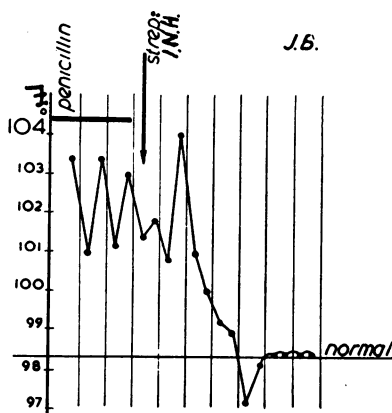


FIG. 4.—Fall in temperature after commencing streptomycin and isoniazid.

and immediate operation at this stage is advisable as the best method of reducing the time of illness. Isoniazid and PAS should be continued for a further three months.

If the glands are soft when first seen antibiotics are certainly not sufficient to prevent further softening and skin involvement, and the pus and caseous material must be removed. Aspiration has been very disappointing even with constant isoniazid and PAS administration, and the shortest period of morbidity occurs when the pus is drained through a small incision and any caseous material present also removed: the wound can either be closed or a small drain left in for two to three days. But, whenever tuberculous glands are seen in this later stage of softening after delayed breakdown, there is always the chance of further abscess formation at the same site or in adjacent glands, and prolonged chemotherapy should be given.

This account of the treatment of tuberculous glands is necessarily incomplete, partly because the disease has such a prolonged natural history, partly because we are still gaining experience with the newer antibiotics, especially isoniazid; it is also because the treatment presents such a wide variety of clinical problems and varying conditions in the glands that my descriptive grouping is in great danger of oversimplification. Yet, even in the acute cases with soft glands and red skin, the cosmetic results are usually much better than first appearance would indicate. Patience, personal care and prolonged antibiotics are probably more important than the precise method of operative technique.

#### REFERENCE

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MILLER, F. J. W., and CASHMAN, J. M. (1955) *Lancet*, **i**, 1286.

#### Mr. Denis Browne:

*Physiology.*—In discussing most systems of the body, such as the nervous, the vascular, or the muscular, it can be taken for granted that their function is understood and that there is a large amount of reasonably accurate information about it. The function of the lymphatic system, apart from the absorption of fats by a limited and specialized portion of it, remains a mystery. The explanations of its workings given in textbooks are either rationalizations of theological outlook . . . "that it wouldn't be there if it hadn't some good purpose" or else due to an almost equally unscientific application of the Darwinian formula of the survival of the fitter. According to this rather inadequate hypothesis only structures which add to the chances of survival, or in other words, have a useful function, are present in the body. This overlooks the fact that some parts, such as the male breast and the toenails, certainly have no function. (It is occasionally necessary to point out that an unproved suggestion that structures may have had a function in different ancestral types is not to give them a function in the present.)

One of the principles of science, in which it differs from politics and theology, is the admission of ignorance. Following this principle we must begin any discussion of such a part of the lymphatic system as the glands of the neck by saying that we are quite ignorant of any purpose they serve, and that we must consider the possibilities that they have no function at all, and are consequently simply a liability, a weak spot in the defences of the body. Supposing, for instance, that all the lymphatic glands of the neck had been removed by dissection, would the resistance of the body to tubercle be any less? If not, what is the meaning in the economy of the body of the infection we are discussing?

*Anatomy.*—Groups of lymph glands drain particular areas, and in these areas there are particular structures which are apt to cause the majority of their infections. In the chest, for instance, the lungs are the main origin of adenitis, and in the abdomen the bowel. Such sources as tubercular skin ulcers or arthritis may occur in the neck as almost anywhere else; but the most common origin of cervical adenitis is the accumulation of lymphatic tissue in the tonsils and adenoids. These strange structures, so vaguely and inaccurately described in books of anatomy and so often inefficiently attacked by surgeons, have never been proved to have any function at all. At the present time it is usually taught that their purpose is to increase the body's resistance to infections which lodge in the ideal bed for multiplication which they provide. Clinically there is no ground whatever to support this hypothesis; chronic infections of the throat cease when they are cleanly removed, and no diminution of general resistance to infection can then be demonstrated.

Though many papers have been published on the subject, it is strange how little tubercular infection of the lymphatic structures of the nasopharynx is realized by the profession at large. I have been present at a discussion of the indications for tonsillectomy at this Society in which tubercle was not mentioned at all by the leading speakers. Another aspect of ignoring this infection is the advice in most textbooks of surgery to obtain an oto-rhino-laryngologist's opinion on whether the tonsils are "infected" in cases of tubercular adenitis; and only then to remove them. As something like 70% of tonsils in such cases show gross tubercle, and as the tubercular tonsil is often small, pale and innocent looking, I do not consider this sound advice.

From this source two distinct forms of infection habitually invade the cervical lymph glands, either separately or in combination. One is tubercle, and the other may be called coccal, since it is usually streptococcal though occasionally staphylococcal. Presumably both infections are either inhaled or swallowed, and gain a first lodging, as diphtheria usually does, in the ideal breeding ground provided by the moist, warm, sheltered crypts of the tonsil.

Consideration of these processes prompts some interesting lines of thought as to the possible connexion between tubercle of the tonsils and tubercle of the lungs. Tuberculous infection of the larynx usually occurs from bacilli passing upwards in sputum, but may also occur from their passage downwards in mucus from the nasopharynx. It is well recognized clinically that coccal infections of the tonsils and adenoids may pass downwards and cause infections of the lungs such as bronchitis. Is it impossible that a similar process may exist with tubercle? I once carried out fifty successive removals of tonsils and adenoids in unselected cases of thoracic tubercle in children. In over 30% of these cases the removed tissue showed gross tuberculous infection, and most striking clinical improvements in the chest condition occurred in many cases; none were harmed. I think more work along these lines is justified. It is occasionally asserted that lymph glands may be infected with tubercle bacilli carried by the blood stream. I know of no proof of this, and as in all cases there is an obvious line of infection along lymphatics I think that this must be very rare if it occurs at all.

The coccal infection typically has an acute or subacute course, in contrast to the slow chronic increase or decline in tuberculous infections. The combination of the two infections often produces a characteristic pattern of illness, with comparatively rapid increases and decreases of coccal origin masking a slow and steady progress of tubercle. The tuberculous infection goes through the well-known stages of simple bacterial invasion, giant cell formation and caseation; but then may take either of two courses. The infection may die out and the gland necrose aseptically, so that finally only unabsorbable calcium salts formed in the caseations remain; or it may form pus which inevitably, if left to itself, discharges through the skin. The tendency to the formation of pus, so much more common in the neck than in the chest or abdomen, seems to have a connexion with secondary coccal infections. Frequently a simple tuberculous adenitis, after remaining hard and unchanged for years, suddenly develops an abscess following a coccal tonsillitis.

Once the discharge of tuberculous pus has started it continues till the necrotic gland tissue at the bottom of the sinus has slowly dissolved; a process that may take years unless shortened by some form of surgery. While the tubercle bacilli in the pus are passing the skin they produce a typical effect upon it, a thinning and final necrosis starting from the deeper layers. The process differs in an interesting way from either tuberculous infection of a skin wound, or from lupus. It resembles tuberculous laryngitis, in that it depends upon the constant contact of tubercle bacilli and ceases as soon as this contact does.

*Diagnosis.*—The early stages of Hodgkin's disease may resemble tuberculous adenitis, and in any cases of doubt a biopsy should be made. It is occasionally difficult to make out whether a subacute coccal infection has an underlying tuberculous element, but on the system of treatment recommended the point is of no clinical importance. An X-ray will often settle the question by showing calcification in the glands.

*Treatment.*—From this description the aims of surgery are obvious. The first is the elimination of the source of the infection, as this can be done without damage to the body as a whole. I was taught many years ago by the late George Waugh that the first step in treating tuberculous cervical adenitis was to remove the tonsils and adenoids without delay, and I still regard this as sound advice.

The second aim is to cut short the long period of discharge from sinuses which is the unassisted reaction of the body. On this aim there are wide differences of opinion and no method guarantees easy and rapid success. The main ones are:

*Excision:* When I first began the study of these cases the popular teaching was that the entire mass of glands should be cut out, preferably before abscesses occurred. This was a severe and difficult operation which at the best left a large scar and a disfiguring flattening of the side of the neck owing to the removal of the subcutaneous tissues. It was also only possible to remove the obviously infected glands, and only too often others showed up later; the argument so often made that excision settled the problem once and for all was far from accurate. In consequence more conservative measures were tried.

*Aspiration:* Aspiration of abscesses was quite ineffective, as it did not remove the necrotic gland and the lumps of caseous material that invariably were present.

*X-rays:* X-rays were sometimes applied, but apart from the well-known dangers of this treatment it had no obvious effect. It must be remembered that subacute coccal infection, already mentioned as being often difficult to distinguish from tuberculosis, subsides spontaneously, and this may often have confused the issue.

*Streptomycin*: Streptomycin seems to have little influence upon an established tuberculous adenitis and for such a comparatively benign condition a full course of this drug is a severe infliction upon a child, apart from its dangers.

*Curettage* of abscesses, either when they have spontaneously discharged or after incision is often satisfactory, but there is the difficulty of knowing the exact location of the necrotic tissue which it is desired to remove.

*Incision and expression* are my own practice, and I think they are the most satisfactory of all. The tonsils and adenoids are removed as soon as possible; if an abscess has already formed there is no objection to combining this operation with its expression. If there is no abscess, nothing is done unless one forms. If this occurs the process is watched till the skin is just adherent over the softening spot, and then an incision a centimetre long is made through which the pus escapes and the necrotic gland is squeezed out of the opening by strong pressure applied by the fingers, with a piece of gauze under them to give a grip and hold the expressed matter. This squeezing is repeated, usually at least a dozen times, till three successive applications of the full force of one's fingers produce nothing but blood. This means that all the necrotic tissue has been expressed, and all that is then necessary is to keep the small opening patent till healing of the large cavity under it is complete. The objection made to this method that it will "spread the infection" is in my experience of several hundred cases quite baseless. The difficulty in getting it applied is that few will use sufficient force and persistence in applying pressure.

If a sinus has persisted for some time there is always contraction of scar tissue along its course, which gives the characteristic indrawn appearance of the skin where it opened. Excision of the scar several years after the infection has subsided, combined with a careful suturing of the subcutaneous fatty tissues, will often make a very marked improvement.

[June 16, 1956]

MEETING IN THE ANATOMY SCHOOL, DOWNING STREET, CAMBRIDGE

## The Decline and Fall in Hospital Pædiatrics

By DOUGLAS GAIRDNER, D.M., F.R.C.P.

In many places in this country, notably in London, the diminishing needs for hospital beds for children have led to a situation where more beds are available than are needed. In the area around Cambridge there has, for various reasons, been an actual shrinkage in the numbers of hospital beds available for children, so that, although the number of sick children requiring hospital care has declined here as elsewhere, there is still some shortage of hospital beds.

This area, with a population of 287,000, comprises Cambridge and those parts of the adjacent counties of Cambs, Hunts, Essex, Herts and Isle of Ely which are either served by the Cambridge group of hospitals, or by hospitals closely linked to them. The area is self-sufficient for all hospital services with the exception of neurosurgery and cardiac surgery, but to balance this receives from outside some pædiatric cases requiring specialist services.

Table I shows the number of hospital beds for children available in the area, and for

TABLE I.—NUMBER OF HOSPITAL BEDS FOR CHILDREN

	Cambridge and surrounding area Pop. 287,000	Newcastle Pop. 295,000
E.N.T. cases .. .. .	26	12
Long-stay cases (tubercle, orthopædic) ..	10	40
All other pædiatric cases, including infectious, but excluding prematures ..	48	110
Total	84	162
Total per 100,000	29	55

comparison, the figures published by Spence and Taylor (1954) for Newcastle for the year 1950. The two areas have about the same population, but differ widely in standards of living—housing, for instance, being relatively good in the Cambridge area, and poor in Newcastle.